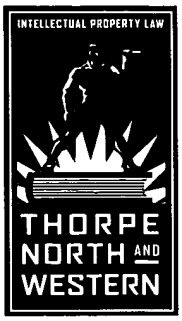
IFW



M. Wayne Western
Registered Patent Attorney
western@tnw.com

Weili Cheng, Ph.D.
Registered Patent Attorney
cheng@tnw.com

October 27, 2004

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

TRANSMITTAL OF INFORMATION DISCLOSURE STATEMENT

Mail Stop Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Re: U.S. Patent Application No. 10/712,073 filed 11/13/2003
Applicant: Beth E. Drees, et al
Title: **LIPID PHOSPHATASE ASSAYS IN DISEASE AND DRUG DISCOVERY**
Group Art Unit: 1641
Attorney Docket No. 22156.NP

Sir/Madam:

Transmitted herewith for filing and pursuant to 37 C.F.R. §§ 1.56 and 1.97 is an Information Disclosure Statement. Enclosed also are the following designated documents, as required under 37 C.F.R. §§ 1.97 and 1.98:

☒ Form PTO-1449 list of 110 references submitted for consideration.

☐ Legible copies of the listed references or their relevant portions.

☒ Legible copies of the listed non-patent documents and foreign documents or their relevant portions are included.

☒ Copies of U.S. patents and/or publications are not included pursuant to the Official Gazette Notice, dated Aug. 5, 2003, waiving the requirement of 37 C.F.R. 1.98(a)(2)(i).

☐ All English translations of each non-English reference, if any, within the possession, custody, control or availability of anyone designated in 37 C.F.R. § 1.56(c) (see 37 C.F.R. § 1.98(c)).

The following are included within the Information Disclosure Statement if applicable and as required under 37 C.F.R. § 1.98:

☐ Concise explanation of relevance of each reference not in English and unaccompanied by an English translation.

☐ Statement that certain listed references not enclosed are substantially cumulative of an enclosed reference.

☐ Statement that certain listed references not enclosed were previously cited by or submitted to the Office in prior application no. , filed on _____, which is relied upon for an earlier filing date under 35 U.S.C. § 120.

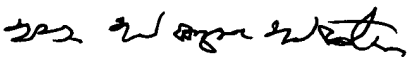
In order to secure consideration of the items designated above, one or more of the following, if required, is also enclosed:

☐ Statement under 37 C.F.R. § 1.97(e)(1) or (2).

☐ Check No. _____, which includes the amount of $0.00 (amount in § 1.17(p)) constituting the submission fee set forth in 37 C.F.R. § 1.17(p).

In the event that 37 C.F.R. § 1.97(c) applies and the Examiner is not satisfied that the Statement meets the requirements of 37 C.F.R. § 1.97(e), or in any other event remediable by a fee, please credit any over payment or charge any additional fees to Deposit Account No. 20-0100 of the undersigned.

Respectfully submitted,



M. Wayne Western
Attorney for Applicant
Registration No. 22,788

THORPE NORTH & WESTERN, LLP
Customer No. 20,551
P.O. Box 1219
Sandy, Utah 84091-1219
Telephone: (801) 566-6633

MWW/WC:ll

H:\FILES\22000\22156\IDSTransmittal.doc

PATENT APPLICATION
ATTORNEY DOCKET NO. 22156.NP

IN THE UNITED STATES PATENT & TRADEMARKS OFFICE

ART UNIT: 1641

EXAMINER:

APPLICANT: Beth E. Drees, et al

SERIAL NO.: 10/712,073

FILED: 11/13/2003

CONFRM. NO.: 7922

FOR: LIPID PHOSPHATASE ASSAYS IN DISEASE AND DRUG DISCOVERY

CERTIFICATE OF MAILING UNDER 37 C.F.R. § 1.8

DATE OF DEPOSIT: 10-28-04

I hereby certify that this paper or fee (along with any paper or fee referred to as being attached or enclosed) is being deposited with the United States Postal Service as first class mail with sufficient postage on the date indicated above and is addressed to: Mail Stop Amendment, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450.

Lucy Leek

Lucy Leek, Secretary

INFORMATION DISCLOSURE STATEMENT UNDER 37 C.F.R. § 1.97

Mail Stop Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir/Madam:

Please find, pursuant to 37 C.F.R. § 1.98(a)(1), the enclosed Form PTO-1449 which contains a list of all patents, publications, or other items that have come to the attention of one or more of the individuals designated in 37 C.F.R. § 1.56(c). Applicant respectfully submits that this Information Disclosure Statement is filed pursuant to:

☒ 37 C.F.R. § 1.97 (b)(1) or (3), within three months of the filing date of the application, or before a first office action on the merits, whichever occurs last;

☐ 37 C.F.R. § 1.97 (c), after a first office action on the merits, but before a Final Office Action or a Notice of Allowance, whichever occurs first, and is accompanied by either 1) a statement in accordance with 37 C.F.R. § 1.97(e), or 2) the fee set forth in§1.17(p); or

☐ 37 C.F.R. § 1.97 (d), after a Final Office Action or Notice of Allowance, whichever occurs first, but on or before payment of the issue fee, and is accompanied by both 1) a statement in accordance with 37 C.F.R. § 1.97(e), and 2) the fee set forth in§1.17(p).

While no representation is made that any of these references may be "prior art" within the meaning of that term in accordance with 35 U.S.C. §§ 102 or 103, the enclosed list of references is disclosed so as to comply with the duty of disclosure set forth in 37 C.F.R. § 1.56.

Moreover, while no representation is made that a specific search of office files or patent office records has been conducted or that no better art exists, the undersigned attorney of record believes that the references listed, together with any other references which may have been previously submitted or listed, are the closest to the claimed invention (taken in its entirety) of which the undersigned is presently aware, and no art which is closer to the claimed invention (taken in its entirety) has been knowingly withheld.

☐ A legible copy of each listed U.S. Patent or publication (or relevant portion thereof) which was not previously submitted to, or cited by, the Patent Office is enclosed pursuant to 37 C.F.R. §§ 1.97 and 1.98.

☒ A legible copy of each of the listed non-patent literature and foreign documents or their relevant portions is enclosed.

☒ Copies of cited U.S. patents and/or publications are NOT enclosed pursuant to the Official Gazette Notice, dated Aug. 5, 2003, waiving the requirement of 37 C.F.R. § 1.98(a)(2)(i).

☐ Copies of the references listed in the accompanying Form PTO-1449 are NOT enclosed because, under 37 C.F.R. § 1.98(d), they were previously cited by or submitted to the Office in application number , which is relied upon for an earlier filing date under 35 U.S.C. § 120.

For all listed references that are not either in the English language, or accompanied by a translation into English, a concise explanation of relevance as required under 37 C.F.R. § 1.98(a)(3) is enclosed attached to each.

The Commissioner is hereby authorized to charge any additional fees associated with this communication or to credit any overpayment to Deposit Account No. 20-0100.

Dated this 28th day of October, 2004.

Respectfully submitted,

M. Wayne Western
Attorney for Applicant
Registration No. 22,788

THORPE NORTH & WESTERN, LLP
Customer No. 20,551
P.O. Box 1219
Sandy, Utah 84091-1219
Telephone: (801) 566-6633

MWW/WC:ll
Enclosures

H:\FILES\22000\22156\IDS.doc

PTO-1449 U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT	APPLICANT Beth E. Drees, et al	
	FILING DATE 11/13/2003	GROUP 1641

U.S. PATENT DOCUMENTS

EXAMINER INITIALS		DOCUMENT NUMBER	DATE	NAME	CLASS	SUBCLASS	FILING DATE IF APPROPRIATE
	A1	2002/0028477	07.03.2002	Goueli et al			
	A2	2003/0100028	29.05.2003	Drees et al			
	A3	5,798,447	25.08.1998	Chen			
	A4	6,001,354	14.12.1999	Pot et al			
	A5	6,348,580	19.02.2002	Fukui et al			
	A6	6,709,833	23.03.2004	Fukui et al			

FOREIGN PATENT DOCUMENTS

EXAMINER INITIALS		DOCUMENT NUMBER	DATE	COUNTRY	CLASS	SUBCLASS	TRANSLATION YES NO
	A7	2001-69977	21.03.2001	JP			
	A8	2000-83664	28.03.2000	JP			
	A9	1-168299 A	03.07.1989	JP			

OTHER PRIOR ART (Including Author, Title, Pertinent Pages, Etc.)

	A10	Martin, T., Phosphoinositide Lipids as Signaling Molecules: Common Themes for Signal Transduction, Cytoskeletal Regulation, and Membrane Trafficking, *Annu Rev Cell Dev Biol,* Vol 14, 231-64 (1998)
	A11	Leslie, N. et al, Phosphoinositide-Regulated Kinases and Phosphoinositide Phosphatases, *Chem Rev*, Vol 101, 2365-2380 (2001)
	A12	Vanhaesebroeck, B. et al, Phosphoinositide 3-kinases: a conserved family of signal transducers, *Trends Biochem Sci*, Vol 22, 267-72 (1997)
	A13	MacDougall, L. et al, A family of phosphoinositide 3-kinases in Drosophila identifies a new mediator of signal transduction, *Current Biology*, Vol 5, No. 12, 1404-15 (1995)
	A14	Carpenter, C. et al, Phosphoinositide 3-kinase and the regulation of cell growth, *Biochimica Biophysica Acta* 1288 (1996) M11-M16
	A15	Carpenter, C. et al, Phosphoinositide Kinases, *Curr Opin Cell Biol*, Vol 8, 153-8 (1996)
	A16	Datta, K. et al, Akt Is a Direct Target of the Phosphatidylinositol 3-Kinase. Activation by Growth Factors, v-src and v-Ha-ras, In Sf9 and Mammalian Cells, *J Biol Chem, Vol* 271, 30835-9 (1996)
	A17	Franke, T. et al, PI3K: Downstream AKTion Blocks Apoptosis, *Cell*, Vol 88, 435-7 (1997)
	A18	Franke, T. et al, The Protein Kinase Encoded by the Akt Proto-Oncogene is a Target of the PDGF-Activated Phosphatidylinositol 3-Kinase, *Cell*, Vol 81, 727-36 (1995)
	A19	Czech, M. et al, Signaling Mechanisms that Regulate Glucose Transport, *J Biol Chem* Vol 274, No. 4, 1865-8 (1999)
	A20	Okada, T. et al, Essential Role of Phosphatidylinositol 3-Kinase in Insulin-induced Glucose Transport and Antilipolysis in Rat Adipocytes, *J Biol Chem* Vol 269, No. 5, 3568-73 (1994)
	A21	Cheatham, B. et al, Phosphatidylinositol 3-Kinase Activation is Required for Insulin Stimulation of pp70 S6 Kinase, DNA Synthesis, and Glucose Transporter Translocation, *Mol Cell Biol*, Vol 14, 4902-11 (1994)

PTO-1449	U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT		APPLICANT Beth E. Drees, et al	
		FILING DATE 11/13/2003	GROUP 1641

	A22	Tanti, J. et al, Potential Role of Protein Kinase B in Glucose Transporter 4 Translocation in Adipocytes, *Endocrinology* 138, 2005-10 (1997)
	A23	Hara, K. et al, 1-Phosphatidylinositol 3-kinase activity is required for insulin-stimulated glucose transport but not for RAS activation in CHO cells, *Proc Natl Acad Sci USA*, Vol 91, 7415-9 (1994)
	A24	Cusi, K. et al, Insulin resistance differentially affects the PI 3-kinase--and MAP kinase--mediated signaling in human muscle, *J Clin Invest*, Vol 105, 311-20 (2000)
	A25	Krook, A. et al, Characterization of Signal Transduction and Glucose Transport in Skeletal Muscle From Type 2 Diabetic Patients, *Diabetes*, Vol 49, 284-92 (2000)
	A26	Cho, H. et al, Insulin Resistance and a Diabetes Mellitus-like Syndrome in Mice Lacking the Protein Kinase Akt2 (PKBβ), *Science*, Vol 292, 1728-31 (2001)
	A27	Krook, A. et al, Insulin-Stimulated Akt Kinase Activity is Reduced in Skeletal Muscle From NIDDM Subjects, *Diabetes*, Vol. 47, 1281-6 (1998)
	A28	Vollenweider, P. et al, An SH2 Domain-Containing 5′ Inositolphosphatase Inhibits Insulin-Induced GLUT4 Translocation and Growth Factor-IInduced Actin Filament Rearrangement, *Mol Cell Biol*, Vol 19, No. 2, 1081-91 (1999)
	A29	Roymans, D. et al, Phosphatidylinositol 3-kinases in tumor progression, *Eur J Biochem*, Vol. 268, 487-98 (2001)
	A30	King, W. et al, Phosphatidylinositol 3-Kinase is Required for Integrin-Stimulated AKT and Raf-1/Mitogen-Activated Protein Kinase Pathway Activation, Mol Cell Biol, Vol 17, No. 8, 4406-18 (1997)
	A31	Kim, D. et al, Akt/PKB promotes cancer cell invasion via increased motility and metalloproteinase production, Faseb J, Vol 15, 1953-62 (2001)
	A32	Phillips, W. et al, Increased Levels of Phosphatidylinositol 3-Kinase Activity in Colorectal Tumors, *American Cancer Society*, Vol 83, 41-7 (1998)
	A33	Klippel, A. et al, Activation of Phosphatidylinositol 3-Kinase is Sufficient for Cell Cycle Entry and Promotes Cellular Changes Characteristic of Oncogenic Transformation, *Mol Cell Biol*, Vol 18, No. 10, 5699-711 (1998)
	A34	Ma, Yen-Ying et al, PIK3CA as an oncogene in cervical cancer, *Oncogene*, Vol 19, 2739-44 (2000)
	A35	Shayesteh, L. et al, PIK3CA is implicated as an oncogene in ovarian cancer, *Nat Genet*, Vol 21, 99-102 (1999)
	A36	Maehama, T. et al, The Tumor Suppressor, PTEN/MMAC1, Dephosphorylates the Lipid Second Messenger, Phosphatidylinositol 3,4,5, Trisphosphate, *J Biol Chem*, Vol 273, 13375-8 (1998)
	A37	Maehama, T. et al, PTEN: a tumour suppressor that functions as a phospholipid phosphatase, *Trends Cell Biol*, Vol. 9, 125-8 (1999)
	A38	Tamura, M. et al, PTEN Interactions with Focal Adhesion Kinase and Suppression of the Extracellular Matrix-dependent Phosphatidylinositol 3-Kinase/Akt Cell Survival Pathway, *J Biol Chem*, Vol 274, 20693-703 (1999)
	A39	Cantley, L. et al, New insights into tumor suppression: PTEN suppresses tumor formation by restraining the phosphoinositide 3-kinase/AKT pathway, *Proc Natl Acad Sci USA*, Vol 96, 4240-5 (1999)
	A40	Haas-Kogan, D. et al, Protein kinase B (PKB/Akt) activity is elevated in glioblastoma cells due to mutation of the tumor suppressor PTEN/MMAC, *Curr Biol*, Vol 8, 1195-8 (1998)
	A41	Wen, S.et al, PTEN controls tumor-induced angiogenesis, *Proc Natl Acad Sci USA*, Vol 98, 4622-7 (2001)
	A42	Li, J. et al, PTEN, a Putative Protein Tyrosine Phosphatase Gene Mutated in Human Brain, Breast, and Prostate Cancer, *Science*, Vol 275, 1943-7 (1997)
	A43	Teng, D. et al, MMAC1/PTEN Mutations in Primary Tumor Specimens and Tumor Cell Lines, *Cancer Res*, Vol 57, 5221-5 (1997)

PTO-1449 U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT	APPLICANT Beth E. Drees, et al	
	FILING DATE 11/13/2003	GROUP 1641

	A44	Dahia, P. et al, PTEN is inversely correlated with the cell survival factor Akt/PKB and is inactivated via multiple mechanisms in haematological malignacies, *Hum Mol Genet*, Vol 8, 185-93 (1999)
	A45	Birck, A. et al, Mutation and Allelic Loss of the PTEN/MMAC1 Gene in Primary and Metastatic Melanoma Biopsies, *J Invest Dermatol*, Vol 114, 277-80 (2000)
	A46	Liaw, D. et al, Germline mutations of the PTEN gene in Cowden disease, an inherited breast and thyroid cancer syndrome, *Nature Genetics*, Vol 16, 64-7 (1997)
	A47	Marsh, D. et al, Allelic Imbalance, Including Deletion of PTEN/MMAC1, at the Cowden Disease Locus on 10q22-23, in Hamartomas From Patients with Cowden Syndrome and Germline PTEN Mutation, *Genes Chromosomes Cancer*, Vol 21, 61-9 (1998)
	A48	Marsh, D. et al, Mutation spectrum and genotype-phenotype analyses in Cowden disease and Bannayan-Zonana syndrome, two hamartoma syndromes with germline PTEN mutation, *Hum Mol Genet*, Vol 7, 507-15 (1998)
	A49	Marsh, D. et al, Germline mutations in PTEN are present in Bannayan-Zonana syndrome, *Nat Genet*, Vol 16, 333-4 (1997)
	A50	Nakashima, N. et al, The Tumor Suppressor PTEN Negatively Regulates Insulin Signaling in 3T3-L1 Adipocytes, *J Biol Chem*, Vol 275, *12889-95* (2000)
	A51	Iida, S. et al, Accelerated Decline of Blood Glucose After Intravenous Glucose Injection in a Patient with Cowden Disease Having a Heterozygous Germline Mutation of the PTEN/MMAC1 Gene, *Anticancer Research*, Vol 20, 1901-4 (2000)
	A52	Butler, M. et al, Specific Inhibition of PTEN Expression Reverses Hyperglycemia in Diabetic Mice, *Diabetes*, Vol 51, 1028-34 (2002)
	A53	Lioubin, M. et al, $p150^{Ship}$, a signal transduction molecule with inositol polyphosphate-5-phosphatase activity, *Genes & Dev*, Vol 10, 1084-95 (1996)
	A54	Damen, J. et al, The 145-kDa protein induced to associate with Shc by multiple cytokines is an inositol tetraphosphate and phosphatidylinositol 3,4,5-trisphosphate 5-phosphatase, *Proc Natl Acad Sci USA*, Vol. 93, 1689-93 (1996)
	A55	Liu, Q. et al, The SH2-Containing Inositol Polyphosphate 5-Phosphatase, Ship, is Expressed During Hematopoiesis and Spermatogenesis, *Blood*, Vol 91, 2753-9 (1998)
	A56	Liu, Q. et al, SHIP is a negative regulator of growth factor receptor-mediated PKB/Akt activationa nd myeloid cell survival, *Genes & Dev*, Vol 13, 786-91 (1999)
	A57	Aman, M. et al, The Inositol Phosphatase SHIP Inhibits Akt/PKB Activation in B Cells, *J Biol Chem*, Vol 273, 33922-8 (1998)
	A58	Helgason, C. et al, Targeted disruption of ShIP leads to hemopoietic perturbations, lung pathology, and a shortened life span, *Genes & Dev*, Vol 12, 1610-20 (1998)
	A59	Brauweiler, A. et al, Differential Regulation of B Cell Development, Activation, and Death by the Src Homology 2 Domain-containing 5' Inositol Phosphatase (SHIP), *J Exp Med*, Vol 191, 1545-54 (2000)
	A60	Liu, Q. et al, The Inositol Polyphosphate 5-Phosphatase Ship Is a Crucial Negative Regulator of B Cell Antigen Receptor Signaling, *J Exp Med*, Vol 188, 1333-42 (1998)
	A61	Rohrschneider, L. et al, Structure, function, and biology of SHIP proteins, *Genes & Dev*, Vol 14, 505-20 (2000)
	A62	Brauweiler, A. et al, Bilevel control of B-cell activation by the inositol 5-phosphatase SHIP, *Immunological Reviews*, Vol 176, 69-74 (2000)
	A63	Bolland, S. et al, SHIP Modulates Immune Receptor Responses by Regulating Membrane Association of Btk, *Immunity*, Vol 8, 509-16 (1998)
	A64	Huber, M. et al, The src homology 2-containing inositol phosphatase (SHIP) is the gatekeeper of mast cell degranulation, *Proc Natl Acad Sci USA*, Vol 95, 11330-5 (1998)

PTO-1449 U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT	APPLICANT Beth E. Drees, et al	
	FILING DATE 11/13/2003	GROUP 1641

	A65	Huber, M. et al, Targeted disruption of SHIP leads to Steel factor-induced degranulation of mast cells, *Embo J*, Vol 17, 7311-9 (1998)
	A66	Pesesse, X. et al, Identification of a Second SH2-Domain-Containing Protein Closely Related to the Phosphatidylinositol Polyphosphate 5-Phosphatase SHIP, *Biochem Biophys Res Commun*, Vol 239, 697-700 (1997)
	A67	Ishihara, H. et al, Molecular Cloning of Rat SH2-Containing Inositol Phosphatase 2 (SHIP2) and Its Role in the Regulation of Insulin Signaling, *Biochem Biophys Res Commun*, Vol 260, 265-72 (1999)
	A68	Muraille, E. et al, Distribution of the Src-homology-2-domain-containing inositol 5-phosphatase SHIP-2 in both non-haemopoietic and haemopoietic cells and possible involvement of SHIP-2 in negative signalling of B-cells, *Biochem J*, Vol 342 Pt 3, 697-705 (1999)
	A69	Habib, T. et al, Growth Factors and Insulin Stimulate Tyrosine Phosphorylation of the 51C/SHIP2 Protein, *J Biol Chem*, Vol 273, 18605-9 (1998)
	A70	Clement, S. et al, The lipid phosphatase SHIP2 controls insulin sensitivity, *Nature*, Vol 409, 92-7 (2001)
	A71	Wada, T. et al, Overexpression of SH2-Containing Inositol Phosphatase 2 Results in Negative Regulation of Insulin-Induced Metabolic Actions in 3T3-L1 Adipocytes via Its 5'-Phosphatase Catalytic Activity, *Mol Cell Biol*, Vol 21, 1633-46 (2001)
	A72	Taylor, G. et al, Myotubularin, a protein tyrosine phosphatase mutated in myotubular myopathy, dephosphorylates the lipid second messenger, phosphatidylinositol 3-phosphate, *Proc Natl Acad Sci USA*, Vol 97, 8910-5 (2000)
	A73	Walker, D. et al, Characterization of MTMR3: an inositol lipid 3-phosphatase with novel substrate specificity, *Current Biology* Vol 11, 1600-5 (2001)
	A74	Maehama, T. et al, A Sensitive Assay for Phosphoinositide Phosphatases, *Anal Biochem*, Vol 279, 248-50 (2000)
	A75	Taylor, G. et al, Myotubularin, a protein tyrosine phosphatase mutated in myotubular myopathy, dephosphorylates the lipid second messenger, phosphatidylinositol 3-phosphate, *PNAS*, Vol 97, 8910-8915 (2000)
	A76	Prestwich, G. et al, In situ detection of phospholipid and phosphoinositide metabolism, *Advan Enzyme Regul*, Vol 42, 19-38 (2002)
	A77	Thomas, C. et al, Generation of phosphatidylinositol-specific antibodies and their characterization, *Biochemical Society Trans,* Vol 27, 648-52 (1999)
	A78	Yokogawa, T. et al, Evidence that 3'-phosphorylated polyphosphoinositides are generated at the nuclear surface: use of immunostaining technique with monoclonal antibodies specific for PI 3,4-P_2, *FEBS Lett*, Vol 473, 222-6 (2000)
	A79	Kavran, J. et al, Specificity and Promiscuity in Phosphoinositide Binding by Pleckstrin Homogoly Domains, *J Biol Chem*, Vol 273, 30497-508 (1998)
	A80	Ferguson, K. et al, Structure of the High Affinity Complex of Inositol Trisphosphate with a Phospholipase C Pleckstrin Homology Domain, *Cell*, Vol 83, 1037-46 (1995)
	A81	Rebecchi, M. et al, Pleckstrin Homology Domains: A Common Fold with Diverse Functions, *Annu Rev Biophys Biomol Struct*, Vol 27, 503-28 (1998)
	A82	Ferguson, K. et al, Structure of the High Affinity Complex of Inositol Trisphosphate with a Phospholipase C Pleckstrin Homology Domain, *Cell*, Vol 83, 1037-46 (1995)
	A83	Stenmark, H. et al, FYVE-finger proteins – effectors of an inositol lipid, *J Cell Science*, Vol 112, 4175-83 (1999)
	A84	Ago, T. et al, The PX Domain as a Novel Phosphoinositide-Binding Module, *Biochem Biophys Res Commun*, Vol 287, 733-8 (2001)
	A85	Dowler, S. et al, Identification of pleckstrin-homology-domain-containing proteins with novel phosphoinositide-binding specificities, *Biochem J*, Vol 351, 19-31 (2000)

PTO-1449 U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT	APPLICANT Beth E. Drees, et al	
	FILING DATE 11/13/2003	GROUP 1641

	A86	Thomas, C. et al, Crystal structure of the phosphatidylinositol 3,4-bisphosphate-binding pleckstrin homology (PH) domain of tandem PH-domain-containing protein 1 (TAPP1): molecular basis of lipid specificity, *Biochem J*, Vol 358, 287-94 (2001)
	A87	Beaudet, L. et al, Homogeneous Assays for Single-Nucleotide Polymorphism Typing Using AlphaScreen, *Genome Res*, Vol 11, 600-8 (2001)
	A88	Latif, S. et al, Fluorescence Polarization in Homogeneous Nucleic Acid Analysis II:5'-Nuclease Assay, *Genome Res*, Vol 11, 436-40 (2001)
	A89	Nielsen, K. et al, Fluorescence Polarization Immunoassay: Detection of Antibody to Brucella abortus, *Methods*, Vol 22, 71-6 (2000)
	A90	Grys, E. et al, Measurement of proteases in human subgingival dental plaque by fluorescence polarization, *Archives Oral Biol*, Vol 45, 1101-6 (2000)
	A91	Economic Consequences of Diabetes Mellitus in the U.S. in 1997, American Diabetes Association. *Diabetes Care*, Vol 21, 296-309 (1998)
	A92	Maehama, T. et al, PTEN and Myotubularin: Novel Phosphoinositide Phosphatases, *Ann Rev Biochem*, Vol 70, 247-79 (2001)
	A93	Bolino et al, Charcot-Marie-Tooth type 4B is caused by mutations in the gene encoding myotubularin-related protein-2, *Nature Genetics*, Vol 25, 17-9 (2000)
	A94	Pope, A. et al, Homogeneous fluorescence readouts for miniaturized high-throughput screening: theory and practice, *Drug Discovery Today*, Vol 4, 350-362 (1999)
	A95	Seifert, R. et al, PTPRQ is a novel phosphatidylinositol phosphatase that can be expressed as a cytoplasmic protein or as a subcellularly localized receptor-like protein, *Exp Cell Res*, Vol 287, 374-386 (2003)
	A96	Ono, H. et al, Regulation of Phosphoinositide Metabolism, Akt Phosphorylation, and Glucose Transport by PTEN (Phosphatase and Tensin Homolog Deleted on Chromosome 10) in 3T3-L1 Adipocytes, *Molecular Endocrinology*, Vol 15, 1411-1422 (2001)
	A97	Mosser, V. et al, PTEN Does Not Modulate GLUT4 Translocation in Rat Adipose Cells under Physiological Conditions, *Biochem Biophys Res Commun*, Vol 288, 1011-1017 (2001)
	A98	Laporte, J. et al, A gene mutated in X-linked myotubular myopathy defines a new putative tyrosine phosphatase family conserved in yeast, *Nature Genetics*, Vol. 13, 175-82, (1996)
	A99	Laporte, J. et al, Characterization of the myotubularin dual specificity phosphatase gene family from yeast to human, *Human Molecular Genetics*, Vol 7, 1703-12 (1998)
	A100	Gray, A. et al, Nonradioactive methods for the assay of phosphoinositide 3-kinases and phosphoinositide phosphatases and selective detection of signaling lipids in cell and tissue extracts, *Analytical Biochem*, Vol 313, 234-245 (2003)
	A101	Ijuin, T. et al, Identification and Characterization of a Novel Inositol Polyphosphate 5-Phosphatase, *J Biol Chem*, Vol 275, 10870-10875 (2000)
	A102	Klarlund. J. et al, Signaling by Phosphoinositide-3, 4,5-Trisphosphate Through Proteins Containing Pleckstrin and Sec7 Homology Domains, Science, Vol 275, 1927-30 (1997)
	A103	Marion, E. et al, The Gene INPPL1, Encoding the Lipid Phosphatase SHIP2, Is a Candidate for Type 2 Diabetes In Rat and Man, *Diabetes*, Vol 51, 2012-17 (2002)
	A104	Buj-Bello, A. et al, The lipid phosphatase myotubularin is essential for skeletal muscle maintenance but not for myogenesis in mice, *Proc Natl Acad Sci USA*, Vol 99, 15060-5 (2002)
	A105	Gaudet, E. et al, A Homogeneous Fluorescence Polarization Assay Adaptable for a Range of Protein Serine/Threonine and Tyrosine Kinases, *J Biomol Screening*, Vol 8, 164-175 (2003)
	A106	Ijuin, T. et al, SKIP Negatively Regulates Insulin-Induced GLUT4 Translocation and Membrane Ruffle Formation, *Mol Cell Biol*, Vol 23, 1209-1220 (2003)

PTO-1449 U.S. DEPARTMENT OF COMMERCE PATENT AND TRADEMARK OFFICE	ATTY. DOCKET NO. 22156.NP	SERIAL NO. 10/712,073
LIST OF PRIOR ART CITED BY APPLICANT	APPLICANT Beth E. Drees, et al	
	FILING DATE 11/13/2003	GROUP 1641

	A107	Kowski, T. et al, Fluorescence Polarization is a Useful Tecnology for Reagent Reduction in Assay Miniaturization, *Comb Chem High Throughput Screen*, Vol 3, 437-44 (2000)
	A108	Blondeau, F. et al, Myotubularin, a phosphatase deficient in myotubular myopathy, acts on phosphatidylinositol 3-kinase and phosphatidylinositol 3-phosphate pathway, *Hum Mol Genet*, Vol 9, 2223-9 (2000)
	A109	Luo, J-M et al, Possible dominant-negative mutation of the SHIP gene in acute myeloid leukemia, *Leukemia*, Vol 17, 1-8 (2003)
	A110	Oganesian, A. et al, Protein tyrosine phosphatase RQ is a phosphatidylinositol phosphatase that can regulate cell survival and proliferation, *Proc Natl Acad Sci USA*, Vol 100, 7563-7568 (2003)

EXAMINER	DATE CONSIDERED

*EXAMINER: Initial if reference considered, whether or not citation is in conformance with MPEP 609; Draw line through citation if not in conformance and not considered. Include copy of this form with next communication with applicant.